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Experimental Research Article

S-Adenosylmethionine protected 5-Fluorouracil-induced cardiotoxicity in male albino rats

Abstract

Introduction: 5-fluorouracil is commonly used for solid cancers, with cardiotoxicity being the main side effect. S-adenosylmethionine (SAMe) is known to have therapeutic benefits in several human diseases, including cancer, osteoarthritis, Alzheimer's disease, depression, and chronic liver diseases. SAMe also possesses cytoprotective and antioxidant properties. The present investigation aims to evaluate the effects of SAMe on 5-Fluorouracil-induced cardiotoxicity in rats by comparing it with a typical medication, Silymarin (SIL).

Methods: Forty male albino rats were divided into five groups as follows: Control (D.W. only), 5-Fluorouracil (100 mg/kg), SAMe (100 mg/kg), 5-Fluorouracil 100 mg/kg+SAMe100 mg/kg, and 5-Fluorouracil 100 mg/kg+200 mg/kg SIL. Cardiotoxicity was induced with an intraperitoneal injection of a single dose of 5-Fluorouracil (100 mg/kg). Serum was collected, and histological analysis of the heart was performed to evaluate the rat model-5-FU's toxicity alone and in combination with SAMe and SIL. In addition to a heart histology examination, serum cardiac enzyme testing, pro-oxidant/antioxidant status, and cyclooxygenase-2 expression in cardiac tissue were examined.

Results: 5-Fluorouracil induced severe cardiotoxicity, as evidenced by increased histological deterioration, cardiac enzymes, cyclooxygenase-2 expression, and malondialdehyde concentrations. The overall antioxidant capacity was likewise reduced by 5-FU treatment. While oxidative stress, cardiac enzymes, histological degenerations, and cyclooxygenase-2 expression in cardiac tissue were reduced, total antioxidant capacity was improved by SAMe or SIL treatment.

Conclusion: The cardiotoxicity induced by 5-fluorouracil in rats was ameliorated by SAMe or SIL therapy confirmed by reserved histological and biochemical analysis.